PEDIATRICS®
OFFICIAL JOURNAL OF THE AMERICAN ACADEMY OF PEDIATRICS

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Pediatrics 1998;102;1494
DOI: 10.1542/peds.102.6.1494

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Misuse of Tanner Puberty Stages to Estimate Chronologic Age

To the Editor.—

One of us has been involved as an expert in several US federal cases of possession of alleged child pornography, in which seized materials (videos, photographs, computer downloads) were used as evidence against individuals identified in "sting" operations, wherein government agents take over pornographic businesses. In these cases the staging of sexual maturation (Tanner stage) has been used not to stage maturation, but to estimate probable chronologic age. This is a wholly illegitimate use of Tanner staging: no equations exist estimating age from stage, and even if they did, the degree of unreliability in the staging—the independent variable—would introduce large errors into the estimation of age, the dependent variable. Furthermore, the unreliability of the stage rating is increased to an unknown degree by improperly performed staging, that is, not at a clinical examination but through nonstandardized and, thus, unsuitable photographs.

Therefore, we wish to caution pediatricians and other physicians to refrain from providing "expert" testimony as to chronologic age based on Tanner staging, which was designed for estimating development or *physiologic age* for medical, educational, and sports purposes—in other words, identifying early and late maturers. The method is appropriate for this, provided chronologic age is known. It is not designed for estimating chronologic age and, therefore, not properly used for this purpose.

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Olanzapine Toxicity in a Toddler

To the Editor.—

Olanzapine (Zyprexa, Eli Lilly) is an atypical antipsychotic approved in 1996 for clinical use in Europe and the United States. There is no recommended pediatric dose for olanzapine or any information on its effect on children.

The mother of a 2½-year-old boy discovered that one or two of her 7.5 mg olanzapine tablets were missing. Within 1 hour the boy was found sleeping and difficult to arouse. Over the next several hours he intermittently awoke. During these times he refused to interact (kiss, speak, play, dance) with family members and remained slow to respond. He was described as glassy-eyed, hostile, agitated, appeared in pain, violent towards family members, and refusing to eat. There was no history of recent illness, head trauma, or seizures. Past medical and surgical history were unremarkable. The child was not on any medications.

The child was evaluated 9.5 hours postingestion because of persistent abnormal behavior. He was a well-nourished, well-developed 13.5-kg male. His blood pressure was 100/60; respiratory rate, 28; heart rate, 120–180; temperature (rectal) 36.9°C; pulse oximetry 98%. HEENT: normal cephalic, atraumatic, pupils 1 to 2 mm and reactive, no nystagmus. Hypersalivation without apparent oropharyngeal lesions was noted. Neck: supple. Lungs: clear with equal breath sounds. Cor: regular tachycardia. Abdomen: normal bowel sounds, soft, and nontender. Neurologic: somnolent and stuporous, yet irritable, agitated, and hostile when aroused. He did not seem to be hallucinating and followed some commands. He was ataxic but muscle tone and reflexes were normal and without clonus.

Complete blood count, electrolytes, anion gap, renal function, urinalysis, head computed tomography scan, and lumbar puncture were normal. Urine toxicology ADX panel was negative for cocaine metabolites, opiates, barbiturates, and benzodiazepines. Serum toxicology 10 hours postingestion was positive for olanzapine by GC-MS: 11 ng/mL (adult therapeutic range 9–23 ng/mL). Electrocardiogram: sinus tachycardia with normal QRS and QT_c intervals. The patient gradually improved to normal during a 24-hour period.

We believe this to be the first report of pediatric olanzapine intoxication. Our patient exhibited agitation, aggressive behavior, miosis, hypersalivation, tachycardia, and ataxia. Adverse effects of olanzapine in adults include somnolence, asthenia, nervousness, insomnia, anxiety, akathisia, tremor, anticholinergic effects, and increased liver function tests.[1] During premarketing trials, overdose of olanzapine was associated with drowsiness and slurred speech.

The onset and duration of clinical effects in our patient were 1 hour and 24 hours, respectively; consistent with olanzapine's peak plasma concentrations (6 hours) and half-life (27–31 hours).[2] Olanzapine is structurally and chemically related to clozapine. The pediatric overdose effects of clozapine are similar in duration (24 hours) and signs and symptoms (ataxia, tachycardia, confusion, hypersalivation, and stupor).[3,4] A prolonged clinical course should be anticipated with olanzapine or clozapine toxicity.

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Selective, Reversible Thalamic Involvement With Influenza A Infection

To the Editor.—

My colleagues and I have read with interest the report by Ruggieri et al[1] regarding selective, reversible thalamic involvement with measles infection. Concerning the etiology of selective, reversible thalamic involvement, the authors concluded that deep cerebral vein thrombosis (DCVT) caused it. We could not deny their hypothesis. However, recently we observed two cases with similar magnetic resonance imaging (MRI) findings and clinical course attributable to influenza A infection. These cases were diagnosed as having the mild form of acute necrotizing encephalopathy (ANE).[2] The authors never mentioned ANE as a possible etiology.

Our cases were previously healthy Japanese boys, aged 2 and 5.[3] Both had acute encephalopathy without pleocytosis in cerebrospinal fluid. Their computed tomography (CT) and MRI showed selective, reversible thalamic involvement and no evidence of DCVT. They recovered without sequelae and disturbance of sensation. Influenza A infection was confirmed serologically in both cases.

It is obvious that influenza A infection also shows selective, reversible thalamic involvement on the MRI, as well as measles infection. Despite the different pathogenic virus, similar mechanisms causing these characteristic MRI findings may be present. The problem is the mechanism. ANE is a novel disease entity